

REMARKS

The final Office Action mailed December 16, 2003, has been received and reviewed. Claims 1 through 45 are currently pending in the application. Claims 1-11 and 16-45 were previously withdrawn. Claims 12-15 stand rejected. Reconsideration is respectfully requested.

35 U.S.C. § 102 Rejections

Claims 12-15 stand rejected under 35 U.S.C. § 102(b) as being anticipated by Borrebaeck (U.S. Patent 6,027,930) (hereinafter "Borrebaeck"). Applicants respectfully traverse this rejection.

In the Office Action, Borrebaeck was thought to disclose "an infectious phage that contains a mutant form of coat protein", which applicants do not concede. (Paper No. 19, page 3). However, that is not the presently claimed invention. Borrebaeck fails to disclose, either expressly or inherently, an infectious phage containing at least one copy of a mutant form of a phage coat protein, **wherein the mutant form** has lost the ability to mediate infection of a natural host by the infectious phage as recited in the pending claims.

Borrebaeck discloses three types of phages. A first phage is a protein-3 deleted helper phage that retains the protein-3 promoter and displays the protein-3 on its surface. (Borrebaeck, col. 5, lines 1-10). The surface protein-3 is provided by a plasmid comprising the sequence encoding protein-3 (*Id.*). The helper phages do not have "at least one copy of a mutant form of a phage coat protein" as recited in claims 12-15.

In contrast to the infectious phage of claims 12-15, Borrebaeck teaches that the mutant coat protein should be complemented with a mutant helper phage resulting in a non-infectious phage. (Borrebaeck, col. 5, lines 28-29). Borrebaeck discloses a second phage produced by cells comprising the identified mutant helper phages and a phagemid encoding for an anti-hen egg lysozyme Fab fragment, fused with the carboxy-terminal part (CT-part) of the protein-3 (Borrebaeck, col. 5, lines 25-29). Thus, in Example 2 of Borrebaeck, the cells containing a pUC19-based plasmid encoding the Fab and CT fusion "were infected with mutant helper phages" (having a genome wherein the nucleotide sequence encoding the p3 protein was deleted). (Borrebaeck, col. 5, lines 28-29, emphasis added). The resulting phages were non-

infectious. (Borrebaeck, col. 5, line 29, emphasis added). As this second phage is non-infectious, it cannot anticipate the infectious phage of claim 12 or the phage collection of claims 13-15.

A third phage of Borrebaeck contains a mutant form of the phage coat protein (a CT/Fab part coupled to a fusion protein containing hen egg lysozyme and a part of the N-terminal part of the protein-3), which “mutant form” **retains** the ability to mediate infection of a natural host by the infectious phage. As the “mutant form” in the third phage retains the ability to mediate infection of a natural host by the infectious phage, it cannot anticipate the infectious phage of claim 12 or the phage collection of claims 13-15 which include the element that the mutant form has lost such an ability.

Borrebaeck is completely focused on the new mutant filamentous helper phage and its high efficiency in SAP technology. (Borrebaeck, Example 3). The SAP technology disclosed in Borrebaeck is based on non-infectious phages having no wild-type protein 3. The selection procedure in the SAP technology is based on the destruction of the basic infectivity of the phage by deleting the N1 domain or the N1 and N2 domains from the gene of protein-3.

In the next step, a peptide or protein library is fused N-terminally to the copies of the CT domain or the N2-CT domains of protein 3. No wild-type protein-3 is present on the phage and the phage is not infectious. The infectivity of the phage can only be restored by adding the N1 or N1-N2 complex. These domains are themselves fused or chemically coupled to a ligand that binds to the peptide or protein displayed on the phage. Only when binding of a ligand to peptide or protein occurs, a protein 3 having all domains necessary for infectivity will be obtained.

When, as suggested by the Examiner, the mutant form of the coat protein would be complemented with a helper phage having protein 3, the SAP technology could no longer be used, because all of the phages would have at least one wild-type protein 3 on their surface and would therefore be infectious. In that case, binding partners could no longer be selected based on restored infectivity.

Thus, Borrebaeck does not disclose, either expressly or inherently, an infectious phage that contains a mutated phage coat protein that has lost the ability to mediate infection of the phage as claimed in the presently claimed invention. Accordingly, Borrebaeck fails to anticipate claims 12-15. Reconsideration and withdrawal of the rejection is requested.

CONCLUSION

Claims 12-15 are believed to be in condition for allowance, and an early notice thereof is respectfully solicited. Should the Office determine that additional issues remain which might be resolved by a telephone conference, the Examiner is respectfully invited to contact applicants' undersigned attorney.

Respectfully submitted,



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